Amendments to the Specification:

Please replace paragraph at page 12, line 30 through page 13, line 9 with the following amended paragraph:

Figure 28 **(a-b)** show the result of identification of nuclear Clusterin (nCLU) as a CCPUCC1-interacting protein by yeast two-hybrid system. FIG. 28(A) shows the interaction of CCPUCC1 with nuclear Clusterin in the yeast cells. FIG. 28(B) shows the interaction between CCPUCC1 and nCLU *in vivo*. COS7 cells were transfected with CCPUCC1-myc or pFlag-Clusterin, or both. Immunoprecipitation was performed with anti-FLAG M2 antibody or anti-myc mouse antibody. Western blot analysis was carried out using anti-myc (upper panel) or anti-FLAG (lower panel) antibody. Bands of CCPUCC1 and C-term nCLU were detected only in the lane of co-transfected cell lysates, which indicates that CCPUCC1 (upper panel) ~~interact~~ **interacts** with nCLU (lower panel) protein *in vivo*.

Please replace paragraph at page 15, lines 19-28 with the following amended paragraph:

Seven genes whose expression levels increased in colonrectal cancers were identified. These seven genes are referred to herein as colon-cancer associated genes. Five of which were novel and two were previously known genes whose association with colon cancer was unknown. The five novel genes include ARHCL1 ("CGX1"), NFXL1 ("CGX2"), C20orf20 ("CGX3"), LEMD1 ("CGX4"), and CCPUCC1 ("CGX5"). The novel colon cancer-associated genes are summarized in Table 1 below and their nucleic acid and polypeptide sequences are provided in the Sequence Listing. The known genes include Ly6E ("CGX6") and Nkd1 ("CGX7"). One known gene, *LAPTM4beta* ("CGX8") whose expression level increased gastric cancer was identified. ~~This~~ **The CGX8** gene is referred to herein as gastric-cancer associated gene.

Please replace paragraph at page 19, lines 20-27 with the following amended paragraph:

Also the expression level of the CGX 1-8 can be analyzed based on the activity or quantity of protein encoded by the gene. A method for determining the quantity of the CGX 1-8 protein is shown in ~~bellow~~ **below**. For example, immunoassay method is useful for the determination of the proteins in biological materials. Any biological materials can be used for the determination of the protein or ~~it's~~ **its** activity. For example, blood sample is analyzed for estimation of the protein encoded by a serum marker. On the other hand, a suitable method can be selected for the determination of the activity of a protein encoded by the CGX 1-8 according to the activity of each protein to be analyzed.

Please replace paragraph at page 28, lines 10-15 with the following amended paragraph:

Alternatively, in another embodiment of the screening method of the present invention, a two-hybrid system utilizing cells may be used ("MATCHMAKER™ Two-Hybrid system", "Mammalian MATCHMAKER™ Two-Hybrid Assay Kit", "MATCHMAKER™ one-Hybrid system" (Clontech); "~~HybriZAP~~ **HYBRIZAP™** Two-Hybrid Vector System" (Stratagene); the references "Dalton and Treisman, Cell 68: 597-612 (1992)", "Fields and Sternglanz, Trends Genet 10: 286-92 (1994)").

Please replace paragraph at page 34, lines 14-20 with the following amended paragraph:

The antisense nucleic acids derivative is given to the patient by directly applying onto the ailing site or by injecting into a blood vessel so that it will reach the site of ailment. Parenteral administration, such as intravenous, subcutaneous, intramuscular, and intraperitoneal delivery routes, may be used to deliver nucleic acids or CGX-inhibitory peptides or non-peptide compounds. An antisense-mounting medium can also be used to increase durability and membrane-permeability. Examples are, liposomes, poly-L-lysine, lipids, cholesterol, ~~lipofectin~~ **LIPOFECTIN™**or derivatives of these.

Please replace paragraph at page 38, line 17 through page 39, line 8 with the following amended paragraph:

Cancer therapies directed at specific molecular alterations that occur in cancer cells have been validated through clinical development and regulatory approval of anti-cancer drugs such as trastuzumab (~~Herceptin~~ HERCEPTIN™) for the treatment of advanced breast cancer, imatinib methylate (~~Gleevec~~ GLEEVEC™) for chronic myeloid leukemia, gefitinib (~~Iressa~~ IRESSA™) for non-small cell lung cancer (NSCLC), and rituximab (anti-CD20 mAb) for B-cell lymphoma and mantle cell lymphoma (Ciardiello F, Tortora G. A novel approach in the treatment of cancer: targeting the epidermal growth factor receptor. Clin Cancer Res. 2001 October;7(10):2958-70. Review.; Slamon D J, Leyland-Jones B, Shak S, Fuchs H, Paton V, Bajamonde A, Fleming T, Eiermann W, Wolter J, Pegram M, Baselga J, Norton L. Use of chemotherapy plus a monoclonal antibody against HER2 for metastatic breast cancer that overexpresses HER2. N Engl J Med. 2001 Mar. 15;344(11):783-92.; Rehwald U, Schulz H, Reiser M, Sieber M, Staak J O, Morschhauser F, Driessen C, Rudiger T, Muller-Hermelink K, Diehl V, Engert A. Treatment of relapsed CD20+ Hodgkin lymphoma with the monoclonal antibody rituximab is effective and well tolerated: results of a phase 2 trial of the German Hodgkin Lymphoma Study Group. Blood. 2003 Jan. 15;101(2):420-424.; Fang G, Kim C N, Perkins CL, Ramadevi N, Winton E, Wittmann S and Bhalla K N. (2000). Blood, 96, 2246-2253.). These drugs are clinically effective and better tolerated than traditional anti-cancer agents because they target only transformed cells. Hence, such drugs not only improve survival and quality of life for cancer patients, but also validate the concept of molecularly targeted cancer therapy. Furthermore, targeted drugs can enhance the efficacy of standard chemotherapy when used in combination with it (Gianni L. (2002). Oncology, 63 Suppl 1, 47-56.; Klejman A, Rushen L, Morrione A, Slupianek A and Skorski T. (2002). Oncogene, 21, 5868-5876.). Therefore, future cancer treatments will probably involve combining conventional drugs with target-specific agents aimed at different characteristics of tumor cells such as angiogenesis and invasiveness.

Please replace paragraph at page 58, lines 17-29 with the following amended paragraph:

Genome-wide cDNA microarray. A genome-wide cDNA microarray with 23040 genes was used. Total RNA extracted from the microdissected tissue was treated with DNase I, amplified with ~~Ampliscribe~~ **AMPLISCRIBE™** T7 Transcription Kit (Epicentre Technologies), and subsequently labeled during reverse transcription with Cy-dye (Amersham). RNA from non-cancerous tissue was labeled with Cy5 and RNA from tumor with Cy3. Hybridization, washing, and detection were carried out as described previously (4), and fluorescence intensity of Cy5 and Cy3 for each target spot was generated by ~~ArrayVision~~ **ARRAYVISION™** software (Amersham Pharmacia). After subtraction of background signal, the duplicate values were averaged for each spot. Then, all fluorescence intensities on a slide were normalized to adjust the mean Cy5 and Cy3 intensities of 52 housekeeping genes for each slide. Genes were excluded from further investigation when the intensities of both Cy3 and Cy5 were below 25,000 fluorescence units, and of the remainder, we selected for further evaluation those with Cy3/Cy5 signal ratios>2.0.

Please replace paragraph at page 59, lines 10-20 with the following amended paragraph:

RNA preparation and RT-PCR. Total RNA was extracted with a Qiagen ~~RNeasy~~ **RNEASY™** kit (Qiagen) or ~~Trizol~~ **TRIZOL™** reagent (Life Technologies, Inc.) according to the manufacturers' protocols. Ten-microgram aliquots of total RNA were reverse transcribed for single-stranded cDNAs using poly dTi.sub.12-18 primer (Amersham Pharmacia Biotech) with ~~Superscript~~ **SUPERSCRIPT™** II reverse transcriptase (Life Technologies). Each single-stranded cDNA preparation was diluted for subsequent PCR amplification by standard RT-PCR experiments carried out in 12-μl volumes of PCR buffer (TAKARA). Amplification proceeded for 4 min at 94°C for denaturing, followed by 21 (for *GAPDH*), 36 (for *ARHCL1*), 32 (for *NFXL1*), 32 (for *C20orf20*), 40 (for *LEMD1*), 30 (for *CCPUCC1*, *Ly6E* and *Nkd1*), and 28 (for *LAPTM4beta*) cycles of 94°C for 30 s, 60°C for 30 s, and 72°C for 60 s, in the GeneAmp PCR system 9700 (Perkin-Elmer, Foster City, Calif). Primer sequences were:

Please replace paragraph at page 62, lines 4-9 with the following amended paragraph:

Effect of anti-sense oligonucleotides on cell growth. Cells plated onto 10-cm dishes ($2X10^5$ cells/dish) were transfected either with plasmid or with synthetic S-oligonucleotides of *ARHCL1*, *NFXL1*, *C20orf20*, *LEMD1*, *CCPUCC1*, *Ly6E*, *Nkd1* or *LAPTM4beta*, using LIPOFECTIN™ Reagent (GIBCO BRL) and cultured for three to seven days. The cells were then fixed with 100% methanol and stained by Giemsa solution. Sequences of the S-oligonucleotides were as follows: